TO STUDY THE CORRELATION OF LIPID LIPOPROTEIN LEVELS WITH STRESS EXERCISE TEST IN ASYMPTOMATIC AND SYMPTOMATIC CORONARY ARTERY DISEASE

THESIS

FOR

DOCTOR OF MEDICINE

(MEDICINE)

D1121

BUNDELKHAND UNIVERSITY

JHANSI (U. P.)

1991

PUSHKIN MEHROTRA

C E R T I F I C A T E

This is to certify that the work entitled "TO STUDY THE CORRELATION OF LIPID LIPOPROTEIN LEVELS WITH STRESS EXERCISE TEST IN ASYMPTOMATIC AND SYMPTOMATIC CORONARY ARTERY DISEASE", which is being submitted as a thesis for M.D.(Medicine) examination, 1991 of Bundelkhand University by Dr. Pushkin Mehrotra, has been carried out in the department of Medicine, M.L.B. Medical College, Jhansi.

He has put in the necessary stay in the department as per university regulations.

29.11.90

Dated: Nov., 1990.

(R. C. Arora)
M.D., D.Sc.,
Professor and Head,
Department of Medicine,
M.L.B. Medical College,
JHANSI.

C E R T I F I C A T E

This is to certify that the work entitled "TO STUDY THE CORRELATION OF LIPID LIPOPROTEIN LEVELS WITH STRESS EXERCISE TEST IN ASYMPTOMATIC AND SYMPTOMATIC CORONARY ARTERY DISEASE", which is being submitted as a thesis for M.D.(Medicine) examination, 1991 of Bundelkhand University by Dr. Pushkin Mehrotra, has been carried out under my direct supervision and guidance. The techniques and statistical methods used were undertaken by the candidate himself and were checked by me from time to time.

Dated: 29.11.90 Nov., 1990.

(R. C. Arora)
M.D., D.Sc.,
Professor and Head,
Department of Medicine,
M.L.B. Medical College,
JHANSI.

(GUIDE)

C E R T I F I C A T E

This is to certify that the work entitled "TO STUDY THE CORRELATION OF LIPID LIPOPROTEIN LEVELS WITH STRESS EXERCISE TEST IN ASYMPTOMATIC AND SYMPTOMATIC CORONARY ARTERY DISEASE", which is being submitted as a thesis for M.D.(Medicine) examination, 1991 of Bundelkhand University by Dr. Pushkin Mehrotra, has been carried out under my direct supervision and guidance. The techniques and statistical methods used were undertaken by the candidate himself and were checked by me from time to time.

29.11.90

Dated : Nov., 1990.

NAgarwal

(Navnit Agarwal)
M.D.,
Lecturer,
Department of Medicine,
M.L.B. Medical College,
JHANSI.

(CO -GUIDE)

CERTIFICATE

This is to certify that the work entitled "TO STUDY THE CORRELATION OF LIPID LIPOPROTEIN LEVEL WITH STRESS EXERCISE TEST IN ASYMPTOMATIC AND SYMPTOMATIC CORONARY ARTERY DISEASE", which is being submitted as a thesis for M.D.(Medicine) examination, 1991 of Bundelkhand University by Dr. Pushkin Mehrotra, has been carried out under my direct supervision and guidance. The techniques and statistical methods used were undertaken by the candidate himself and were checked by me from time to time.

29.11.90

Dated: Nov., 1990.

(Praveen Kumar)
M.D., Dip.Card., D.M.(Card.),
Lecturer in Cardiology,
M.L.B. Medical College,
JHANSI.

(CO-GUIDE)

C E R T I F I C A T E

This is to certify that the work entitled "TO STUDY THE CORRELATION OF LIPID LIPOPROTEIN LEVELS WITH STRESS EXERCISE TEST IN ASYMPTOMATIC AND SYMPTOMATIC CORONARY ARTERY DISEASE", which is being submitted as a thesis for M.D.(Medicine) examination, 1991 of Bundelkhand University by Dr. Pushkin Mehrotra, has been carried out under my direct supervision and guidance. The techniques and statistical methods used were undertaken by the candidate himself and were checked by me from time to time.

29.11.90

Dated: Nov., 1990.

(Sunita Arora)
M.S.,
Reader,
Obstetrics and Gynaecology,
Department,
M.L.B. Medical College,
JHANSI.

(CO-GUIDE)

A C K N O W L E D G E M E N T

The most pleasant duty in a work like this is to remember and ruminate my obligations to all those who have made its completion possible.

I consider it a privilege to express with a deep sense of gratitude my indebtedness to my Chief Guide Prof. R.C. Arora, MD, MCCP, FICA, D.Sc., Head, Department of Medicine, M.L.B. Medical College, Jhansi for the untiring help, able guidance, astute and unfailing scruitiny and careful supervision provided by him during the entire period of this study. It would be no exaggeration to state that but for his endless patience, invaluable suggestions and his dedication to provide me with every opportunity to learn the subject, this endeavour would never have seen light. Things that I have learnt from him will take me a long way in my career. He has not only been a valuable and immense help in my thesis but a memory that would be everlasting.

Words fail to express my deepest sense of gratitude and sincere most thanks to my respected Coguide Dr. Navnit Agarwal, M.D., Lecturer in Medicine, M.L.B. Medical College, Jhansi, who ungrudgingly rendered his valued criticism and learned advice as and when neede till completion of this work. His efforts to get me over my initial hesitations and

oinspire in to me a sense of confidence were overwhelming. His cheerful attitude and reassuring presence in helping me tide over my difficulty faced in the work shall remain in my thoughts for ever.

My sincere most thanks are due to Dr. (Mrs.) Sunita Arora, M.S., Reader, Deparrment of Obst. & Gynaec., M.L.B. Medical College, Jhansi who always helped me in every possible way to achieve my target. Her valuable suggestions, constructive criticism and meticulous attention have gone a long way towards the success of this work.

In no less degree I owe my sincere most thanks to my Co-guide Dr. Praveen Kumar, M.D., D. Card., D.M., (Card.), Lecturer in Cardiology, M.L.B. Medical College, Jhansi with his store of unlimited knowledge and excellence in expression, this work would be brought out in its present form.

I find myself indebted to Prof. D.N. Mishra, MD,MNAMS, FCCP, Professor of Medicine, Dr. G.D. Shukla, MD,MNAMS, Ph.D., Lecturer in Psychiatry, Dr. P.K. Jain, MD,MNAMS, Lecturer in Medicine, Dr. T.V.S. Arya, MD, Lecturer in Medicine for providing a constant source of inspiration and enthusiasm to complete the present work.

I will be failing in my duty if I do not extend my sincere most thank to Dr. Nirbhai Kumar, M.D., Senior Resident, Department of Medicine for his

invaluable, meticulous and consistent help throughout the period of this study. In no less degree I thank to my junior colleagues.

I also owe my thanks to my wife who provided moral support and encouragement during my hours of desparation due to overwhelming problems and time consuming process.

I am also thankful to Mr. Phool Chandra Sachan for bringing out such a neatly typed script.

Lastly, I thank to all the subjects who volunteered in this project.

Dated: 29.11.90.

Mehrotra

(Pushkin Mehrotra)

CONTENT

INTRODUCTION

INTRODUCTION

Atherosclerosis remains one of the leading cause of morbidity and mortality due to cardiovascular diseases. Several factors are known to be associated with premature atherosclerosis. Among them the most important factors are hypertension, elevated serum cholesterol, smoking, obesity, diabetes mellitus, stress and strain, sedentary life style and prolonged family history. Abnormal lipid levels may be common to several of risk factors.

Researchers are unsure about the exact cause of atherosclerosis and also about the most effective methods of intervention. Diseases such as diabetas and hypertension require medication for control but most of other risk factors can be modified by dietary changes and an increase in physical activity.

Atherosclerosis, leading to C.V.D. begins early in life. It has been argued that the physicians cannot adequately detect and manage the children at risk of CVD. Data from multiple epidemiological studies of risk factors of CVD suggest that the risk factors can be identified in children and that they demonstrate a "Tracking Phenomenon" into adult life. Many of risk factors are largely determined or influenced by life style or environmental factors. There are no prospective studies that show a

relationship between the presence of risk factors of C.V.D. in children and premature cardiovascular disease in later life.

The age of patient and the life situation in which treatment is initiated are of obvious importance. The coronary artery disease (CAD) has familial association, both because of genetic predisposition and nutritinal imbalance which develops in a family setting. So, children should be included in this consideration of possible preventive treatment for two reasons, first, atherosclerosis has its beginning in childhood and is continuously progressive throughout the life. Second, the patterns of food consumption are largely developed early in life.

In five studies, between 18-25 percent of the progeney of parents who sustained myocardial infarction before the age of 55 years showed significant hypercholesterolemia and/or hypertriglyceridemia in (Glueck, 1983), while in control group the incidence of hyperlipidemia stands 6 percent. Few other studies reported that sons (14-25 years) of fathers with ischemia had lower levels of HDL(Nupuf and Sutherland, 1979), a protective factor for CHD, then controls.

It seems that there may be a subset even within the genetically predisposed group, who are more likely to develop CHD prematurely by virtue of their unfavourable

lipid profile. This identification can possibly done by estimating their basal lipid profile or by a provocative test comprising, a high fat diet, high cholesterol load, for short duration.

Several studies have shown that a significant number of patients of CAD have abnormal fasting and/or lipid lipoprotein profile but the exact co-ordination has never been worked out. In the proposed study we have tried to show this relationship, we have evaluated whether patients of CAD(Documented by Positive Exercise Test) possess abnormality in their lipid-lypoprotein profile. For labelling a case as CAD we adopted the time honoured method exercise ECG, if resting ECG was normal. The wide spread use of exercise testing during routine annual examinations has defined a hereto fore unrecognised group of patients with asymptomatic CAD. In addition, patients who are asymptomatic after an infarct are none the less at greater risk for a second CAD event than the general population. Although medical therapy (Elimination of smoking, anti-hypertensive medications) aimed at preventing progression of the disease is indicated, recent surgical data suggesting that by pass surgery in prove mortality in coronary patients. While such an approach cannot be condoned as the basis of available data, a number of factors should be considered :

(1) The degree of positivity of the exercise test and duration or stage of exercise at which it appears.

(2) The ECG leads in which the test is positive (changes in the anterior precordial leads appears to indicate less favourable prognosis than the changes in the inferior leads).

(3) The age of the patients.

(4) The occupation of patients where as most would agree that the asymptomatic, 45 years old, commercial air like-pilot with 4 mm S^T segment depression in leads $V_1 - V_4$ during mild exercise should have arteriogram and the asymptomatic sedentary, 75 years old retired with 1 mm ST segment depression in leads II and III during maximal exercise should not, there is no consensus about the appropriate procedure in less extreme situations.

At present time ECG stress testing is the best approximation of this ideal because electrocardiographic ST segment displacement is a characteristic response of to ischemia and occurs during and immediately following an exercise test. Similarly, the false negative responses are known to occur and may be due to the complex geometry of the heart, these limitations notwithstanding, the realization that SE segmant depression is a clinically useful, readily available index of ischemia has prompted the development of standardized exercise tests which are ST segment depression as the end point to define ischemia.

The ischemic ST segment response is generally defined as a flat depression of ST segment 0.1 mV below the base line, i.e. PR segment lasting more than 0.08 seconds. If 0.1 mV or more ST segment depression is required before the test is considered to be positive, the percentage of false positive results will be less than 10 percent and only about 15 percent of patients with severe coronary atherosclerosis will have negative test results.

The duration of exercise, presence of anginal symptoms, heart rate and blood pressure response are also important in evaluating the results of a stress electro-cardiogram. Thus, false positive results are least likely in those patients who demonstrate ST segment depression early in the test, in whom the changes persist for relatively long period (5 minutes) following cessation of exercise, who experience typical angina during the test.

Approximately 10 percent of patients with a positive test will have ST segment changes only during exercise. Exercise testing is safer within exercise monitoring because ST segment changes or arrhythmias may develop before pain or other symptoms occur. Three formats of stress testing are commonly used; tests with standardised external work loads, test which are standardised by heart rate response, and tests designed to reach the

maximal possible exercise load. The two step system of exercise developed by Master is an example of the first variety. The second and third varieties employ a bicycle exercise test. In the target heart rate test, exercise is continued until the patient attains 80-90 percent of his predicted maximum heart rate. In the maximal exercise test is progressively increased until maximal work load is obtained.

R E V I E W   O F   L I T E R A T U R E

REVIEW OF LITERATURE

The majority of people below the age of 55 years are afflicted with atherosclerosis and have one or more identifiable risk factors other than aging per se. The risk factor concept implies that a person with at least one risk factor is more likely to develop a clinical atherosclerotic event and to do so earlier than a person with no risk factors. The presence of multiple risk factors further accelerates atherosclerosis. They vary in terms of importance in the population. There is general agrement from an epidemiologic perspective that hypercholesterolemia, hypertension and cigarette smoking may be the most potent factors involved in the causation of atherosclerosis. Risk factors also vary in terms of their potential reversibility with current techniques of preventive management.

Thus, age, sex and genetic factors are currently considered to be irreversible risk factors, whereas continually emerging evidence suggests that elimination of cigarette smoking and treatment of hypertension reverses high risk of atherosclerosis attributable to these factors.

These factors are not mutually exclusive and they clearly interact. For example, obesity appears to be casually associated with hypertension, hyper-

glycemia, hypercholesterolemia and hypertriglyceridemia. Genetic factor may play a role by exerting direct affects on arterial wall structure and metabolism or they may act indirectly via such factors as hypertension, hyperlipidemia, diabetes and obesity. Aging appears to be one of the more complex factors associated with the development of atherosclerosis, since many of risk factors in themselves are related to aging, e.g. elevated blood pressure, hyperglycemia and hyperlipidemia.

HISTORICAL ASPECTS OF ATHEROSCLEROSIS

Atherosclerosis has been recognised in humans for thousand of years. Lesions of atherosclerosis were identified in Egyptian mummies as early as the fifteenth century B.C. Long has discussed the development of clinical pathological correlations that evolved during the era when autopsy examination permitted the development of an understanding between the degree of atherosclerosis and the incidence of myocardial infarction and stroke. In the mid nineteenth century, Virchow proposed the idea that some form of injury to the artery wall associated with an inflammatory response resulted in what was then considered to be a degenerative lesion of atherosclerosis. This idea was subsequently modified by Anitschkow and further included the role of platelets and thrombogenesis in atherosclerosis as expanded by Duguid in 1948. Many of

modern views of atherosclerosis stem from the work of John French who noted that the structural integrity of the endothelial lining of the artery represented a key element in the maintenance of normal arterial function and that alterations in endothelial lining of the artery represented a key element in the maintenance of normal arterial function and that alterations in endothelial integrity might precede a sequence of events that leads to the various forms of the lesion of atherosclerosis. Thus over the ye ars a number of theories concerning the etiology and pathogenesis of atherosclerosis have been developed. At least three of those deserve elaboration and comment. These are response to injury hypothesis, monoclonal hypothesis and the lipogenic hypothesis.

THE LESIONS OF ATHEROSCLEROSIS

Examination of atherosclerotic lesions with modern techniques of cell and molecular biology has revealed that each lesion contains significant element of all the three cellular phenomenon. These are smooth muscle proliferation, formation by the proliferated cells of large amounts of connective tissue matrix including collagen, elastic fibres and proteoglycans and accumulation of intracellular and extra cellular lipid. In each instance, the relative degree to which each of the cells responds to different atherogenic stimuli determines the unique combination that defines the type

and the extent of the resulting lesion (Rose and Glomset, 1976).

The lesions of atherosclerosis occur principally within the inner most layer of the artery wall, the intima. They include fatty streaks, the fibrous plaque and so called complicated lesions (Mc Gill, 1977). Secondary changes have been noted in the media of the artery underlying lesions, principally in association with the more advanced lesions of atherosclerosis.

THE FATTY STREAK

The process of atherosclerosis begins in the childhood with the development of flat, lipid rich lesions called fatty streaks. These lesions consist of a small increase in the number of smooth muscle cells together with some macrophages within the arterial intima. Both of these cell types contain deposits of cholesterol and cholesterol esters. The lesions are yellowish and sessile in appearance and cause little to no obstruction of the affected artery and no clinical sequelae.

THE FIBROUS PLAQUES

The fibrous plaque is grossly white in appearance and becomes elevated so that it may protrude into the lumen of artery. If this lesion progresses sufficiently it can occlude the lumen and compromise the vascular supply of the involved tissue. The principal change that occurs within the arterial intima during the

development of the fibrous plaque consists of proliferation of smooth muscle cells. These cells usually form a fibrous cap due to deposition by the cells of new connective tissue matrix and the accumulation of intracellular and extracellular lipids. This fibrous cap covers a deeper deposit of varying amounts of extracellular lipid and cell debris (Ceer and Haust, 1972).

ADVANCED LESION

The complicated lesions of atherosclerosis occur in increased frequency with increasing age. The fibrous plaque can become vascularised both from the luminal as well as medial aspects. In the complicated lesion, the necrotic "lipid rich core" increases in size and often becomes calcified. The lesions may become increasingly complex as a result of haemorrhage and calcification and the intimal surface may disintegrate and ulcerate and become involved with thrombotic episodes that may lead to occlusive disease. Such thrombi may then organize and further increase the thickness of the plaque while progressively reducing the size of the arterial lumen. It is not uncommon that as the intimal lesions progress, the number of smooth muscle cell in the underlying media decreases and media undergoes atrophy, which can sometimes results in aneurysmal changes rather than lead to thrombotic occlusion of artery.

Ideal lipoprotein values.

LDL	$<$100 mg/dl*
HDL	$>$ 50 mg/dl*
LDL/HDL (ratio)**	$<$ 2.0
Triglycerides	$<$150 mg/dl+

* These numbers are based on values found in very very low risk groups from population in segeral countries (The lipid research clinics Population studies, Data Book, volume, I, 1980; Conference on Health effects of blood lipids, 1979).

** The ratio of LDL/HDL is a very good index of risk (Gordon et al., 1977). Thus, with levels of HDL greater than 50, higher levels of LDL may be associated with low incidence of vascular disease.

\+ The triglyceride value is arbitrary. It represwnts the value two standard deviations above the mean for an adult population (conference on Health effects of blood lipids, 1979). Higher values are thought to represent a group of metabolic abnormalities. This may have no relationship to vascular disease in presence of below average total plasma cholesterol ($<$200 mg/dl) or "ideal" HDL ($>$50 mg/dl).

In addition, HDLs serve as acceptors of lipid, especially free cholesterol from various tissues. HDLs are the substrate for Lecithin : cholesterol acyltransferase (L-CAT), which catalysis the conversion of both free cholesterol to cholesterol ester and lecithin to lysolecithin. Cholesterol esters are transferred from HDLs to other lipoproteins nonspecifically as well as by a cholesterol-ester transfer protein. This process provides core constituents for triglyceride depleted particles such as chylomicron remnanats. Apolipoproteins A-I and A-II are major proteins of HDLs. Hepatic lipase is involved in the metabolism of HDL phospholipids and triglycerides. Both the liver and the kidney appear to be major sites of HDL catabolism, and HDL receptor has been reported to exist in a variety of cell types.

EFFECTS OF DIETARY CHOLESTEROL IN LIPID METABOLISM

For 70 years the addition of dietary cholesterol has been known to increase plasma cholesterol levels and induce arteriosclerosis in experimental animals. In 1912 it was identified as the constituent of animal foods which would readily elevate the serum cholesterol level and produce atherosclerosis in experimental animals. Subsequently cholesterol rich diets have regularly caused hypercholesterolemia, atherosclerosis, and even at times myocardial infarction in a large number of species of

experimental animals, including primates (Taylor et al, 1950; Armstrong et al, 1967 and 1970). For a time,however, dietary cholesterol was considered of little importance in human lipid metabolism. By the early 1960s, a decisive effect of cholesterol in the diet of man upon the serum lipid levels was clearly demonstrated in series of metabolic ward experiments being carried out in normal columteers (Conner et al, 1961; Conner et al, 1964 and Beveridge et al., 1960).

Dietary cholesterol is absorbed by the gut in amounts proportional to intake up to a dietary level of perhaps ≅1200 to 1500 mg/day. Only about 40 percent is absorbed (Conner and Lin, 1974; Grundy et al, 1969).

In man, absorbed cholesterol is transported initially in chylomicrons, largely as esterified cholesterol and reaches a peak concentration in plasma 48 hours after a given meal. After the action of lipoprotein lipase in peripheral tissues, it then circulates as cholesterol rich remants before removed by Liver. The cholesterol of remnants contributes its mass to the total body pools of cholesterol(Bhattacharya et al, 1976).

There is good evidence that cholesterol of dietary origin in transferred ultimately into other lipoprotein classes, especially in low density lipoprotein (LDL) and contributes in this manner to elevations of total plasma cholesterol (Conner and Conner, 1977).

The increased plasma cholesterol was transported chiefly in LDL for the normal and hypercholes-

terolemic subjects. Slight increases occurred in HDL. In hypertriglyceridemic subjects however, both VLDL and LDL cholesterol increased, each accounting for about 50 percent of total increase.

Most of the dietary cholesterol is quickly delivered to the liver where several effects may be observed. The increased cholesterol uptake may :

1. Inhibit new cholesterol synthesis.
2. Increase sterol excretion in the bile as bile acids or as cholesterol itself.
3. Increase excretion of cholesterol from the liver as newly synthesized lipoproteins primary VLDL, or
4. Suppress specific receptors for LDL uptake and degradation.

This leads us to a consideration of the mechanism whereby dietary cholesterol increased the total plasma cholesterol concentrations and also LDL cholesterol concentrations. The concepts of sterol balance suggests that dietary cholesterol may simple overload the disposal system. The input of sterols into the plasma tissue pool has two sources, from dietary cholesterol and from cholesterol synthesized mainly by the liver and gut. The rate of cholesterol synthesis in man is not very labile, most studies have shown a mild depression of synthesis from the ingestion of dietary cholesterol (Lin and Conner, 1981). Therefore, the total amount of sterol entering the body either from diet or by synthesis will be much greater in

individuals consuming a high cholesterol diet than in individuals consuming low cholesterol diet. Most studies to date have indicated that the bile acid and neutral steroid excretion fails to increase very much after ingestion of dietary cholesterol. Thus the ingestion of large quantities of dietary cholesterol may have two consequences. The first is a rise in plasma cholesterol and LDL concentrations. The second, a direct result of the first, is the ultimate deposition of an increased amount of cholesterol in tissues, particularly in arteries, to initiate and sustain the atherosclerotic process.

DIETARY LIPIDS AND HDL

Much research has indicated the importance of specific dietary components in modulating levels of serum lipids, but there is yet little information regarding the effects of these components on HDL. In short term feeding studies, marked reduction in dietary fat and isocaloric increase in carbohydrate resulted in a decrease in HDL cholesterol in conjuction with elevation of serum triglyceride and VLDL. Studies of HDL composition have shown a decrease in ratio of apolipoprotein A-I to A-II and a decrease in HDL cholesterol to protein ratio (Schonfeld et al, 1976) consistent with a selective decrease in HDL_2 species (Blum et al., 1977).

There is evidence that substitution of large quantities of polyunsaturated fat for saturated fat in diet can result in lower levels of HDL lipids and

proteins (Nichman et al, 1967). An increase in the P:S ratio from 0.25 : 1 to 4 : 1 in real food diets fed to four normal subjects for five weeks resulted in reductions, of HDL cholesterol and apolipoproteins A-I concentration of 33 and 21 percent respectively, with an associated reduction in HDL_2 : HDL_3 ratio (Shepherd et al, 1978). Other studies have however, reported either no change (Lewis, 1978; Shore et al, 1981) or increases (Jackson and Glueck, 1980) in levels of HDL cholesterol with feeding of diets enriched in polyunsaturated fat.

High dietary intake of cholesterol, in the form of three to six egg yolks per day, has been reported to produce increases in apolipoprotein E - containing HDL sub-species in humans (Mahley et al, 1978). This effect was seen whether or not there was an increase in total plasma cholesterol. Despite the fact that HDL containing apo-lipoprotein E represented only a minor fraction of the total HDL, its presence was shown to account for an increase of 2.6 to four times the binding of HDL to LDL receptors of fibroblasts as compared to pretreatment HDL (Mahley et al, 1981). But this was not observed in another study (Applebaum et al, 1979). Recently it has been reported that levels of HDL cholesterol and serum apolipoprotein A-I, but not apolipoprotein E increased with the feeding of diets high in both cholesterol and saturated fat (Tan et al, 1974). As described earlier an increase in ratio of cholesterol to triglyceride in

HDL has been observed during the feeding of 750 mg of added dietary cholesterol in normal subjects (Mistry et al, 1977).

HABITUAL DIET AND CORONARY ARTERY DISEASE

Although diet is not usually considered a primary risk factor of cardiovascular disease, it has been associated with several other risk factors of cardiovascular disease, including abnormal lipid lipoprotein levels, hypertension, diabetes mellitus and obesity. Although the effects of consuming excessive sodium, sugar and total calories on hypertension, diabetes and obesity are well documented, the influence of diet on lipid and lipoprotein levels, and eventually on cardiovascular disease is still controversial (Stambler, 1978).

The Oslo heart study, a randomized controlled trial of primary CHD prevention among middle aged men at high CHD risk has reported favourable and significant results in regard to CHD incidence and mortality based on reduced intake of saturated fat and cholesterol(without recommendation of high polyunsaturated fat intake).

DIETARY HABITS AND BLOOD LIPID LIPOPROTEIN

In adolescents with initial cholesterol levels greater than 200 mg/dl, a 50 percent decrease in cholesterol intake led to an appreciable drop (15.6%) in cholesterol levels, but the effects was much more modest (8.3%) in those with lower intial levels (Mc Gandy et al, 1972). In a large survey of school children, there was

no positive correlation between the low (80 to 130 mg/dl) the intermediate (157 to 170 mg/dl) and the high (194 to 426 mg/dl) cholesterol levels, with the mean daily intake of energy, sugar, fat, saturated fat and cholesterol (Weidman et al, 1978). However, in 7 different studies summarized recently, significantly albeit weak, correlations were noted between serum lipids and dietary P/S ratio of fat, cholesterol, protein, carbohydrate and sucrose (Mellies and Glueck, 1983). In a survey of school age children examining the influence of nutrients on HDL and LDL cholesterol, it was concluded that the higher intake of cholesterol and lower ratio of P/S was associated with higher values of LDL cholesterol. Larger intake of carbohydrates led to decreased HDL cholesterol and increased triglycerides (Khoury et al, 1980; Laskarzewski et al, 1979). Despite the relatively low magnitude of the partial correlation coefficients between dietary factors and serum lipids in children, it is still possible to conclude that nutrient intake of calories and fat play a small but significant role relative to serum lipids and lipoproteins (Mellies and Glueck, 1983).

GENETIC FACTORS OF CORONARY HEART DISEASE

The genetic aspects of coronary heart disease (CHD) have been extensively evaluated. Familial clustering CHD strongly suggests that genetic factors play an important role in etiology (Deutscher et al, 1970;

Epstein, 1964; Rose, 1960). Some studies suggest that familial aggregation of CHD may be influenced both by genetic characteristics of various risk factors.

FAMILIAL HYPERCHOLESTEROLEMIA

Of the diseases producing hypercholesterolemia in man, familial hypercholesterolemia is the best defined clinically genetically and biochemically. The disorder, results from one of several genetic defects in a cell surface receptor that normally controls the degradation of low density lipoprotein (LDL). Familial hypercholesterolemia is characterized by three cardinal features.

1. Selective elevation in the plasma level of LDL.
2. Deposition of LDL derived cholesterol in abnormal sites in the body, especially in tendons (forming xanthomas) in arteries (forming atheromas) and
3. Inheritance as an autosomal dominant trait with a gene dosage effect, that is, individuals inheriting two mutant allelles (homozygotes) are more severely affected than those inheriting on mutant allele (heterozygotes).

Familial hypercholesterolemia was first genetic disorder recognised to cause myocardial infarction. To this day, it remains the outstanding example of a single gene mutation that causes both hypercholesturolemia and coronary atherosclerosis.

Heterozygous with familial hypercholesterolemia occur at a frequency of about 1 in 500 persons, whereas homozygotes constitute 1 in one million persons population.

Heterozygotes can be diagnosed at birth because blood plasma from the umbilical cord contains a two to three fold increase in the concentration of LDL cholesterol. The elevated levels of plasma LDL persist through out life, but symptoms typically do not develop until the third or fourth decade. The most important clinical feature is premature and accelerated atherosclerosis. Myocardial infarction begin to occur in affected men in the third decade, showing a peak incidence in fourth or fifth decades. By age 60, approximately 80 percent have experienced a myocardial infarction.

Homozygotes have marked elevation in the plasma level of LDL from birth. Coronary artery atherosclerosis frequently has its clinical onset in homozygotes before age 10, and myocardial infarction has been reported as early as 18 months of age.

ELEVATED LIPID LIPOPROTEIN LEVELS
AND RISK OF ATHEROSCLEROSIS

Cholesterol

Elevated total cholesterol is a risk factor for coronary heart disease. As the level rises above 180 mg/dl the risk of developing CHD increases. A cholesterol value of 220 mg/dl represents a nearly two

fold elevation in the incidence when compared with level of 180 mg/dl (Kannel et al, 1971).

Likewise, patients with angiographically defined CHD have significantly higher cholesterol concentrations then patients without CHD, with increased levels associated with a greater number of diseased vessels (Cohn et al, 1977).

LDL Cholesterol

LDL-C which approximately 75 percent of total serum cholesterol may be more specifically associated with coronary artery disease than is total cholesterol.

It has been known for many years that the reduction of elevated LDL in other primate species is followed by regression of arteriosclerotic lesions in coronary arteries in larger vessels (St. Clair, 1983). We have now conclusive evidence in humans that reducing elevated LDL cholesterol will reduce the incidence of clinical events attributable to coronary arteriosclerosis (The lipid research clinics coronary primary prevention trial results, 1984).

HDL Cholesterol

HDL levels have an inverse relationship with coronary artery disease (Gordon et al, 1977). The ability of HDL cholesterol to predict the developing of coronary atherosclerosis has been estimated to be four times

greater than LDL cholesterol and eight times greater than total cholesterol (Gordon et al, 1977). Each 10 mg/dl change in HDL cholesterol concentration with 50 percent alteration in cardiovascular risk(Brensike,et al, 1984).

Sub classes of HDL can be fractioned by zonal ultracentrifugation and include HDL_2 and HDL_3. Among these subgroups HDL_2 appears to have the strongest inverse relationship with coronary artery disease and accounts for different levels of HDL-c between men and women(Gofman et al., 1954). The possible mechanism by which HDL cholesterol decreases atherosclerosis include :

1. Reversal of cholesterol transport from the pheripheral cells to the liver for removal from the body (Miller and Miller, 1975).

2. Inhibition of LDL cholesterol uptake by cells at the LDL receptor sites.

__Triglycerides__

The level of plasma, triglycerides, reflecting increased levels of VLDL also predict increased cardiovascular risk (Carlson et al, 1979 and Kannel et al, (1979). However, there is currently great debate as to whether VLDL is direct operative factor in producing vascular disease, or whether it is the association of increased LDL or decreased HDL levels which are causative (Bilheimer et al, 1972 and Berman et al, 1978). Thus VLDL triglyceride may only be a marker for other lipoprotein abnormalities.

LIPOPROTEINS : PATHOGENIC ROLE

Low density lipoproteins and intermediate density lipopeoteins enter the arterial intima from plasma in man at rates directly related to their plasma concentrations (Neihaus et al, 1977; Nicoll et al, 1981) and accumulate particularly in regions already atheromatous. Endothelial injury greatly enhances this process, and may itself be caused experimentally by raised concentration of plasma lipids (Ross and Harker, 1976). The cholesterol of atheromatous lesions is principally derived from plasma (Zilversmit, 1963). The interactions of LDL with cells of atheromatous plaques have been studied in some detail. Smooth muscle cells and fibroblasts have receptors that mediate uptake of LDL(Goldstein and Brown, 1974 and Bierman and Albers, 1975), its cholesterol is released by lysosomal degradation. Macrophages lack these receptors but acquire lipoprotein cholesterol by other processes, including receptor mediated uptake of altered LDL. In contact with cultural mendothelial cells, LDL is modified, permitting macrophages to degrade it (Henriksen et al, 1981).

At the higher tissue concentrations resulting from pronounced hyperlipidemia substantial amounts of LDL enter macrophages and other cells independently of receptors - by fluid phase endocytosis and other process (Steinberg, 1981). The foam cells of atheromatous plaques and xanthomas result from accumulation of lipoprotein cholesterol from peripheral cells to the liver, it may

lipids by these processes and their degeneration leads to extracellular lipid deposits of atherosclerosis (Small, 1977).

OTHER MECHANISMS

These, then, are some of the mechanisms underlying the association between high concentrations of LDL, coronary heart disease and atherosclerosis. LDL may also contribute to other aspects of atherosclerosis by further actions. LDL, particularly light LDL in man and in animals fed cholesterol, is selectively mitogenic to arterial smooth muscle (Fless et al, 1982). Studies on cultured cells suggest that LDL and other mitogens derived from platelets and endothelium might lead to the smooth muscle hyperplasia of atheromatous plaques.

Most cells meet their requirements for cholesterol (for membrane synthesis) synthesis chiefly by uptake of plasma LDL and other lipoproteins, local synthesis of cholesterol is a further source in some tissues. Except for cells that synthesize lipoproteins and those using cholesterol for steroid harmone synthesis cells in the steady state must possess other mechanisms for releasing cholesterol at rates equal to their uptake and synthesis. Cultured cells show a net efflux of cholesterol (Fielding and Fielding, 1982). As the liver is the only organ that excrets cholesterol, mechanisms must exist for reverse, centripetal transport of cholesterol cholesterol from peripheral cells to the liver, it was

suggested in 1968 that HDL participates in centripetal transport. Soon after Miller and Miller (1975) advanced the concept that this function of HDL by favouring mobilization of cholesterol from arterial well, might explain the inverse srelation between HDL and risk of coronary heart disease.

THE LIPOPROTEINS : PREDICTORS OF CHD ?

LDL and HDL together carry more than 90 percent of the cholesterol in plasma. Independent but interacting antagonistic but closely associated, they are the "Odd couple" of plasma. The epidemiological aspects of their associations with coronary heart disease have been studied in depth, these associations are strong, predictive and independent of other risk factors as we expect of casual relations.

Concentrations of LDL cholesterol are directly related to and are predictive of the risk of coronary heart disease over a wide age range (Gordon et al, 1981). This underlies the association between coronary heart disease and serum cholesterol, for the latter reflects LDL concentrations (Kannel et al, 1979). Mortality rates from coronary heart disease in different communities are directly and linearly related with serum concentrations of cholesterol and LDL cholesterol (Lewis et al, 1978). HDL cholesterol concentrations are even more stongly predictive of the risk of coronary heart disease in most

(Gordon et al, 1981) and Goldbourt and Medalie, 1979) but not all (Wiklund et al, 1980), studies, the relation being inverse; but unlike LDL, HDL cholesterol concentrations do not correlate inversely with mortality rates from coronary heart disease in different countries.

Hyperlipidemia as well as other risk factors run in families and have a tendency to "track" or maintain their rank overtime. Screening for hypercholesterolemia at age of 12 years, is fairly predictive of adult hypercholesterolemia close to 50 percent of the top quintile (88%) for cholesterol, were similarly placed at follow-up nine years later. Of interest was the observation that those who dropped out the top quintile at follow-up had a lower incidence of obesity, smoked less and were more active (Orchard et al, 1983).

Predictions from measurements of total cholesterol can be improved upon by measuring HDL cholesterol, which is known to be protective against CHD and contributes proportionately more to the total cholesterol concentration at childhood. In a survey of 6775 school children, a substantial proportion of those with hypercholesterolemia were attributable to HDL cholesterol levels (Morrison et al, 1979). A recent study examining the influence of family history of CHD, hypertension, obesity and diabetes on total cholesterol, HDL cholesterol and LDL cholesterol, concluded that, in view of

their variation with age, screening during adolescence permitted a more accurate identification of individuals likely to become "high risk" adults (DuRant et al,1982).

More precise estimation of risk can be obtained from samples of LDL and HDL cholesterol. The ratio of total cholesterol (reflecting LDL) to HDL cholesterol is about as efficient as any other lipid profile (Kannel et al., 1979). A ratio of 5 indicates the average high risk in affluent western populations, and ratios exceeding this are a definite cause of concern within the range of serum cholesterol values that are commonly encountered. A more optimal ratio is in the vicinity of 3.5 corresponding to half the standard risk and resembling that found in low CHD in incidence countries (Gordon et , al., 1982).

MATERIAL AND METHODS

MATERIAL AND METHODS

Case material for the present study consisted of 32 male subjects aged between 40 to 60 years, who were divided into following groups :

GROUP I

This group consisted of 10 healthy subjects. These subjects were subjected to single dose high cholesterol fat diet (HCFD) and on the basis of post prandial (vide infra) were again divided into two subgroups IA and IB.

Group IA

This group consisted of individual who showed adverse lipid profile on single dose HCFD and were labelled as "High risk group" (10 subjects). These subjects showed a significant rise of STC and/or HDL at first post prandial hour with or without fall of HDL.

Group IB

This group was similarly assessed and on same criteria were labelled as "Low risk group" (4 subjects). These subjects showed fall of STC and/or HDL with or without rise of HDL.

GROUP II

This group had 18 subjects who were subdivided in two groups IIA and IIB.

Group IIA

All (9) cases of group IIA documented coronary artery disease (CAD) on electrocardiogram and clinical criteria.

Group IIB

This group consisted of 9 subjects. They were asymptomatic with normal resting ECG but revealed abnormality in their ECG's on exercise.

Subjects of group I were asked to stop all medicines (interfering with interpretation of ECG 3 weeks prior to the study). On the day of study subjects were asked to come with light breakfast and were allowed to rest for half an hour. The Blood pressure was recorded in both supine as well as standing posture. After which a 12 lead ECG was recorded in next step. They were subjected to exercise on bicycle Ergometer on graded speeds to achieve 85% of target heart rate(THR), test was stopped when any of following occurred, (1) when THR was achieved, (2) when patients felt chest pain or tiredness, (3) when obvious ischemic changes were recorded.

Simultaneous recording of blood pressure was recorded through out the exercise.

Subjects of group II were asked to have an overnight fast of 14 hours. Next day morning fasting

blood sample was drawn in recumbent posture without producing venous stasis (Koerselmer et al, 1961). HCFD consisting of 3 eggs plus 250 ml of milk was given and post prandial samples were taken at first and third postprandial hour.

Detailed past history related to the episode of myocardial infarction, present complaint, if any, dietary and personal history were also recorded. Through out the procedure subjects were confined to bed and were not allowed to smoke. Serum was separated from each blood sample and following lipid parameters were assessed by enzymatic kit method :

1. Serum total cholesterol (STC).
2. High density lipoprotein (HDL)
3. Serum Triglyceride (STG).

Low density lipoprotein (LDL) was calculated with the help of following formulae given by Fredrickson, 1972.

$$LDL = STC - (STG/5 + HDL) \text{ mg/dl.}$$

A I M S   A N D   O B J E C T S

AIMS AND OBJECTIVES

Studies related with single and prolonged high cholesterol fat diet loading in healthy volunteers and its effects on serum lipid profile are being done in our department for the last so many years. The proposed study has been planned to assess whether some of these subjects who were at high risk (as per their fasting and post prandial lipid profile) actually had increased risk of CAD. The study also aims at detecting any abnormality in lipid profile(both fasting as well as postprandial) in proved cases of CAD. Thus the aims of the present study were :

1. To assess the incidence and severity of IHD by resting and exercise tests in subjects who were fed single dose of high cholesterol fat diet (HCFD).
2. To find out incidence of abnormal cholesterol test in proved cases of coronary artery disease.

O B S E R V A T I O N S

OBSERVATIONS

The present study was carried out in 32 male subjects aged 40 to 60 years. These subjects were divided into 4 following groups as was described in section 3.

GROUP IA

It consisted of 10 healthy males in age group of 45-60 years with mean age of 44.7 ± 7.2 years and mean weight of 61.9 ± 3.9 kg. Their general characteristics are shown in table 1.

GROUP IB

It consisted of 4 healthy males in age group of 45-50 years with mean age of 44.7 ± 1.9 and mean weight of 61.2 ± 1.9 kg. Their general characteristics are shown in table 1.

GROUP IIA

It consisted of 9 patients of documented coronary artery disease with myocardial infarction in age group from 40 to 60 years. The mean age 50.7 ± 7.8 years and mean weight was 60.4 ± 1.4 kg. Their general characteristics are shown in table 2.

GROUP IIB

It consisted of 9 healthy male subjects with age group of 40-60 years with mean age of 48.1 ± 3.4 years and mean weight of 60.7 ± 2.0 kgs. Their general characteristics are shown in table 2.

SOME GENERAL CHARACTERISTICS OF GROUP I

Sl. No.	General Characteristics	Group IA No.	Group IA %	Group IB No.	Group IB %
1.	**OCCUPATION**				
	a. Class IV employees	9	90	4	100
	b. Business class	-	-	-	-
	c. Doctors	1	10	-	-
	d. Engineer	-	-	-	-
2.	**PHYSICAL ACTIVITY**				
	a. Sedentary	1	10	-	-
	b. Moderate	-	-	-	-
	c. Active	9	90	4	100
3.	**FAT CONSUMPTION**				
	a. Low fat consumption ($\angle$40 gm of visible fat/day).	6	60	4	100
	b. High fat consumption (40 or more than 40 gm of visible fat/day).	4	40	-	-
4.	**DIETARY HABIT**				
	a. Vegetarians	7	70	4	100
	b. Non-vegetarian	3	30	-	-
5.	**SMOKING**				
	a. Smokers	9	90	4	100
	b. Non-smokers	1	10	-	-

SOME GENERAL CHARACTERISTICS OF GROUP II

Sl. No.	General Characteristics	Group IIA No.	Group IIA %	Group IIB No.	Group IIB %
1.	OCCUPATION				
	a. Class IV employees	9	100	8	89
	b. Business class	-	-	-	-
	c. Doctor	-	-	-	-
	d. Engineer	-	-	1	100
2.	PHYSICAL ACTIVITY				
	a. Sedentary	9	100	9	100
	b. Moderate	-	-	-	-
	c. Active	-	-	-	-
3.	FAT CONSUMPTIONS				
	a. Low fat consumptions (Less than 40 gm/of visible fat per day.	4	55	6	67
	b. High fat consumption (40 or more than 40 gm of visible fat/day).	4	44	3	33
4.	DIETARY HABIT				
	a. Vegetarian	5	55	6	67
	b. Non-vegetarian	4	44	3	33
5.	Smoking				
	a. Smokers	4	44	6	67
	b. Non-smokers	5	55	3	33

CHANGES IN GROUP IA

Subjects in this group (N=10) were labelled as high risk as per their post prandial lipid profile. These subjects were fed single dose HCFD sometime between 1984-1989. All except 1(10%) had fasting STC below 200 mg%. The mean fasting STC in this group was 163.4±30.3 mg%. All these subjects were taken from high risk group on the basis of postprandial values. STC increased at one hour in all of them. The mean STC value at one hour was 205.8 ±43.2 mg%. The value at third hour postprandial hour approximated the fasting value. The third hour value was 193.6±25.3 mg%. The statistical analysis shows that increase at first hour was significant (Table 3).

TABLE 3 : Showing changes in total serum cholesterol (STC) after single dose HCFD(Mean±SD mg%).

Fasting (I)	1 hour (II)	3 hour(III)
163.4±30.3	205.8±43.2	183.6±25.3
I : II	't' = 2.51	$p < 0.05$
I : III	't' = 1.60	$p > 0.05$

The maximum increase in STC was 117 mg%(90% of fasting in subject No. 6) while the minimum increase was 6 mg%(2.7% of fasting value in subject No. 8).

The mean fasting LDL was 82.7±34.2 mg%. It increased at one hour after HCFD to a mean level of 120±35.6 mg%. The value at third hour approached

fasting value as has been shown in table 4. The rise of LDL at first postprandial hour was significant.

TABLE 4 : Depicting changes in LDL level in group IA (Mean±S.D. mg%).

Fasting (I)	1 hour(II)	3 hour(III)
82.7±34.2	120.0±35.6	95.6±23.6
I : II	't' = 2.36	$p < 0.05$

The maximum increase in LDL was 61 mg%(109% of basal value - subject No. 6), while minimum increase was 6 mg%(3.6% of basal value in subject No. 4). The value of HDL did not show much difference on feeding as has been shown in table 5.

TABLE 5 : Showing changes in HDL on HCFD feeding. (Mean ±S.D. mg%).

Fasting (I)	1 hour(II)	3 hour(III)
65.8±18.3	69.2±23.6	58.4±11.2

The values when compared were found statistically insignificant.

The level of serum triglyceride (STG) did not show any significant change at first postprandial hour, but it increased at third hour postprandial though this increase was statistically insignificant as shown in table 6.

TABLE 6 : Changes in STG level in group IA. (Mean ±S.D. mg%).

Fasting (I)	1 hour(II)	3 hour(III)
124.1±43.4	122.3±44.08	150.6±40.2
I : III	't' = 1.40	p $>$0.05

STRESS TEST AND OTHER PARAMETERS

All subjects in this group had normal ECG on rest. When subjected to exercise 2 of them subject No. 8 and 10 showed changes in ST segment in the form of depression of 2.5 and 2 mm respectively. The depression was in the form of down sloping.

The mean resting heart rate in this group of subjects was 83.0±15.6/min. The heart rate on peak exercise reached to a mean level of 160±17.4/min. The maximum increase in HR was recorded in subject No. 2 (94/min an increase of 127% over resting value). The minimum increase was in subject No. 8, was 66/min. (66% of resting value). This subject was one of the two positive responders to stress test. Other subject No. 10 showed an increase of 82/min. (95% increase over resting value).

The mean systolic blood pressure on rest was 128.6±12.2 mm Hg. It increased in all the subjects. The mean systolic BP at peak of exercise was 156.8± 16.6 mmHg. This rise is systolic BP was statistically significant (Table 7).

TABLE 7 : Showing effect of exercise on heart rate and blood pressure in group Ia subjects. (Mean$\pm$S.D.)

Resting HR (I)		HR on peak exercise (II).
83.0$\pm$15.6		160.0$\pm$17.4
I : II	't' = 10.32	$p < 0.001$
Resting systolic BP(I)		Systolic BP on peak Exercise(II)
128.6$\pm$12.2		156.8$\pm$16.6
I : II	't' = 4.28,	$p < 0.01$
Resting diastolic BP(I)		Diastolic BP on peak exercise(II)
78.8$\pm$5.8		87.4$\pm$6.3
I : II	't' = 3.14	$p < 0.01$

The maximum increase in systolic BP was in subject No. 5(60 mm Hg; 46% over resting value), while the minimum increase was in subject No. 1(10 mm Hg, 7% over resting value).

The subject No.8 who showed positive response had an increment in systolic BP of 30 mm Hg (21% of resting value). The other subject (No. 10) showed a rise of 32 mm Hg (23% of resting value).

The mean diastolic BP at rest was 78.8$\pm$5.8 mm Hg. It reached to 87.4$\pm$6.3 mm Hg at peak of exercise. All except one (10%) subject showed increase in their diastolic BP. One subject did not show any change in diastolic BP even on maximum exercise.

The maximum increase in diastolic BP was in subject No. 6(16 mm Hg, 21% over resting value) while there was no change in diastolic BP in subject No. 5. The positive responders to stress test No. 8 and 10 showed an increase of 4 mm Hg (5% over resting value) and 10 mm Hg(11% of resting value) respectively.

CORRELATION OF POSITIVE RESPONSE ON STRESS TEST AND ABNORMAL LIPID LIPOPROTEIN PROFILE

Subject No. 8 show showed positive response on stress test had minimum increase in STC on HCFD feeding (6 mg%, 2.7% of fasting value) of all subjects. Interestingly he had maximum level of fasting STC (215 mg%). The other positive case (Subject No. 10) showed an increase of 12 mg% (9% of fasting value). This subject had the fasting STC well within normal range(130 mg%, one of the lowest values in this group).

LDL

Subject No. 8 and 10 both showed increase in their LDL values - 58 mg%(61% of basal value) and 17.4 mg%(44% of basal value on HCFD feeding).

Thus both the positive cases in this group showed increase in their STC and LDL level HCFD feeding, though this increase was not maximum in this group.

INCIDENCE OF ABNORMAL STRESS ECG IN SUBJECTS WITH HIGH AND LOW RISK

Out of total 10 healthy subjects who showed abnormal lipid profile on HCFD feeding(abnormal choles-

terol tolerance) only 2 had abnormality in their stress ECG (ischaemic changes) while subjects with low risk showed no abnormality in stress ECG. No subject of any group showed any abnormality in resting ECG. Thus 20% of healthy subjects who were having abnormal cholesterol tolerance showed ischaemic changes in ECG.

CHANGES IN GROUP IB

TABLE 8 : Changes in lipid lipoprotein profile. (Mean ±S.D. mg%).

Parameters	Fasting (I)	1 hour(II)	3 hour(III)
STC	148.7±27.8	124.5±22.9	133.0±22.9
I : II	't' = 1.402		$p > 0.05$
HDL	48.2±11.9	46.2±9.2	41.5±9.9
I : II	't' = 0.248		$p > 0.01$
STG	130.0±3.6	142.0±24.8	137.0±13.4
I : II	't' = 0.895		$p > 0.05$
LDL	76.7±22.2	57.5±7.6	62.1±13.6
I : II	't' = 1.53		$p > 0.05$

The volunteers in this group were among the low risk group as per their post prandial behaviour i.e. they all showed a fall in STC and LDL on HCFB feeding with or without rise of HDL.

I(a). CHANGES IN STC LEVEL

The mean STC in this group of subject was 148.7±29.8 mg%. All the subjects in this group showed

a fall in STC at first post prandial hour, though this fall was statistically insignificant as is evident from table 8. The third hour value increased but wal well below the fasting value in all the 4 subjects. This change in third hour value when compared to fasting value was found to be again insignificant.

The maximum fall of STC was found in subject No. 4 who showed a fall of 45 mg%(23% of the basal value). while the minimum decrease was recorded in subject No.3. It was a fall of 11 mg%, which amounted to a fall of mere than 7% of basal value.

I(b) CHANGES IN HDL LEVEL

HDL level in this group of subjects also did not show any significant change after feeding of test diet. The fasting level (48.2±11.9 mg%) decreased slightly to 46.2±9.2 mg% at first post prandial hour and fell again to 41.5±9.9 mg% at third hour. These changes, however, were statistically insignificant as has been shown in table 8.

The maximum fall of HDL was observed in subject No. 4(7 mg%, 10% of fasting value), while one subject No. 2 showed no change at first hour. Subject No. 3 showed an increase in HDL, rather than decrease, though this increase was merely of 2 mg% (5% of basal value).

I(c). CHANGES IN STG LEVEL

Two subjects (50%) showed a rise in STG at one hour, 1(25%) subject showed fall, while there was no change in STG in 1(25%) subject.

The mean STG in this group of subjects was 130.0±3.6 mg%. It increased to a level of 142.0±24.8 mg% at first hour. The mean value at third hour again showed a fall (137.0±13.4 mg%). All these changes when were statistically analysed were found to be insignificant(Table 8).

The maximum rise of 50 mg%(37.5%) of basal value) was observed in subject No. 3, while one subject in this study group (subject No. 2) showed a fall of 14 mg%(10.7% of basal value). Subject No. 2 showed no change. Thus this group exhibited an inconsistent postprandial behaviour as far as STG was concerned.

I(d). CHANGES IN LDL LEVEL

LDL followed the same postprandial behaviour changes as were observed with STC. All subjects showed fall at first hour. This value increased to fasting value at third postprandial hour in all the subjects. except one(25%). The latter subject showed a further fall at third hour (subject No. 1).

The mean LDL in this group of subjects was 76.7±22.2 mg%. It fell to 57.5±7.6 mg% at first hour. The value at third post prandial hour was 62.1±13.6 mg%.

These all changes were statistically insignificant (Table 8).

The maximum fall of 39 mg% (36% of basal value) subject No. 4 and the minimum fall of 17 mg%(25%) of basal value; subject No. 2) were observed. One subject, as mentioned earlier (subject No. 1) showed a rise of 3 mg% (6% of basal value) at first hour.

ELECTROCARDIOGRAPHIC AND HAEMODYNAMIC CHANGES IN GROUP IB

All subjects in this group had normal resting as well as exercise ECG recordings.

TABLE 9 : Showing effect of exercise on HR and BP in group IB.(Mean$\pm$S.D.).

Resting value (I)	Value at peak exercise(II)
Heart rate/min	
82.0$\pm$9.6	158.0$\pm$8.4
I : II 't' = 11.4, $p < 0.001$ (highly significant).	
Systolic BP mm Hg(I)	
120.0$\pm$7.07	150.0$\pm$12.4
I : II 't' = 3.94, $p < 0.01$ (Significant)	
Diastolic BP mm Hg	
73.5$\pm$6.06	88.0$\pm$2.0
I : II 't' = 4.28, $p < 0.01$ (Significant).	

The mean resting heart rate in this group of subjects was 82.0$\pm$9.6/min. It increased to 158$\pm$8.4/min.

at peak exercise. This increase was statistically highly significant. The maximum rise in heart rate was observed in subject No. 1(94/min, 138% of resting value). The minimum rise was in subject No. 2. It was 58/min. amounting to an increase of 63% over resting value.

All subjects in this group had normal resting systolic as well as diastolic BP at rest.

The mean systolic BP in this group was 120$\pm$7.0 mm Hg. All subjects showed a rise on exercise On peak exercise this value reached to 150$\pm$12.4 mm Hg. This change was statistically significant (Table 9). The maximum rise of systolic blood pressure was observed in subject No. 5(50 mm Hg, 41% over resting value). The minimum rise of systolic blood pressure was 18 mm Hg (13.8% over resting value, subject No. 3).

The diastolic blood pressure increased in all the subjects of this group. The mean diastolic blood pressure was 73.5$\pm$6.0 mm Hg. It was increased to 88.0$\pm$2.0 mm Hg on peak exercise. This change was statistically significant. The maximum rise was observed in subjbect No. 2(20 mm Hg; 28.5% over basal value), while the minimum rise was of 6 mm Hg in subject No. 4, amounting to 7%of resting value.

All subjects in this group had normal lipid profile and also exhibited a normal resting as well as stress ECG recordings.

COMPARISON OF HAEMODYNAMIC PARAMETERS BETWEEN GROUP IA AND IB

The mean resting heart rate in group IA (subjects of high risk) was 83.0±15.6/min, while it was 82.0±9.6/min. in group IB(subjects with low risk). Thus the mean heart rate in these two study groups were virtually similar (t = 0.115) statistically insignificant. The mean systolic BP in group IA was 128.6±12.2 mm Hg. The same parameter was 120.0±7.0 mm Hg in group IB. The difference in blood pressures were statistically insignificant (t=1.280, p $>$0.05). The mean diastolic blood pressure in high risk group (IA) was 78.8±5.8 mm Hg. This value was 73.5±6.0 mm Hg in low risk group (IB). The difference again was statistically insignificant (t = 1.477, p $>$0.05).

CHANGES IN GROUP IIA

TABLE 10 : Showing type of myocardial infarction(MI) (wall involved) in patients of group IIA.

Sl. No.	Wall Involved	No.of cases	Percentage
1.	Inferior wall	2	22.00
2.	Antero-septal wall	3	34.00
3.	Antero-lateral wall	2	22.00
4.	Ext.-anterior wall	1	11.00
5.	Antero-lateral and inferior wall	1	11.00

This group had patients of established myocardial infarction (documented by resting ECG).

The cardiac wall involved in these patients are shown in table 10.

CHANGES IN LIPID LIPOPROTEIN PROFILE

IIa) : CHANGES IN STC

This group of subjects showed a variable response in STC after the first dose of HCFD. Six subjects (67%) showed increase in STC level at first hour after feeding while remaining 3(33%) showed fall. The resting STC was above 200 mg% in 5(56%) subjects. It was below 200 mg% in 3(33%) subjects, while one(11%) had the value of 200 mg%.

The mean STC level in this group of subjects was 211.6±32.6 mg%. At first postprandial hour it increased to 218.4±23.5 mg%. It showed a little fall at third hour (215.0±25.6 mg%). All the values when statistically analysed were found to be insignificant as is shown in table 11.

TABLE 11 : Table depicting changes in STC after HCFD in group IIA. (mean±S.D. mg%).

Fasting (I)	1 hour (II)	3 hour(III)
211.6±32.6	218.4±23.55	215.0±25.6
I : II	't' = 0.494	
I : III	't' = 0.239	

Out of six subjects who showed a rise in STC the maximum rise was observed in subject No. 6(26 mg%, 13% of basal value), while minimum rise was 8 mg%

(4% of basal value, subject No. 9). The 3 subjects who showed a fall after HCFD (subjects No. 3,4, & 5). The maximum fall of 20 mg% amounting to 7% of fasting value was observed in subject No. 5. The minimum fall was of 6 mg%(2.5% of fasting value, subject No. 4).

I(b): CHANGES IN HDL

Six subjects showed rise of HDL after feeding while 3 showed a fall. The mean HDL in this group of subjects was 51.7±8.2 mg%(fasting value). It increased to 55.3±11.8 mg% at first post prandial hour. The level approached the fasting level at third hour (53.5±11.8 mg%). All these changes were statistically insignificant shown in table No. 12.

TABLE 12 : Changes in HDL level after HCFD in group IIA (mean ± S.D. mg%).

Fasting (I)	1 hour (II)	3 hour (III)
51.7±8.2	55.3±11.8	53.5±11.8
I : II	't' = 0.732	
I : III	't' = 0.366	

The maximum rise of HDL was in subject No. 1. It was 16 mg% amounting to 4% of fasting value. The minimum rise was seen in subject No. 3(4 mg%, 7% of basal value). The maximum fall in HDL was observed in subject No. 4(10 mg%, 27% of basal value). The minimum fall of 6 mg%,(27% of basal value). The minimum fall

of 6 mg% was observed in subjects (No. 5 and 6). It amounted to 10.7% of basal value, in both the subjects as basal values, too, were similar.

I(c) : CHANGES IN STG

All subjects showed rise in STG level after test diet. The mean fasting STG level in this group was 171.7±17.7 mg%. It increased to 188.5±17.4 mg% at first hour. This rise was statistically significant as shown in table 13.

TABLE 13 : Depicting changes in STG after HCFD in group IIA(Mean±S.D. mg%).

Fasting (I)	1 hour (II)	3 hour (III)
171.7±17.7	188.5±17.4	185.7±17.2

I : II 't' = 2.049, $p < 0.05$ (Significant)

I : III 't' = 1.729

The values at third hour fell slightly to 185.7±17.2 mg%. This value when compared with fasting value, was found to be statistivally insignificant. The maximum rise of 30% mg% was observed in subject No. 2 amounting to 17.6% of fasting value. The minimum rise was seen in subject No. 4(10 mg%, 5.6% of basal value).

I (d) : CHANGES IN LDL

Five subjects of group IIA showed rise in LDL level after ingestion of test diet while remaining four showed a fall. The mean fasting LDL level was 125.5±32

mg%. It remained all most same at first hour but fall to a level of 102.2±24.3 mg% at third hour. This fall was statistically insignificant as shown in table 14.

TABLE 14 : Showing changes in LDL after HCFD in group IIA(Mean±S.D. mg%).

Fasting (I)		1 hour(II)	3 hour(III)
125.6±32.0		125.5±28.8	102.2±24.3
I : II	't' = 0.0036.		
I : III	't' = 1.70,	p > 0.05(insignificant).	

The maximum rise of LDL was observed in subject No. 6(27.6 mg%, 24.4% of basal value). The minimum rise was seen in subject No. 4(2 mg%, 1.2% of fasting value). The maximum fall was seen in subject No. 3(20.8 mg%, 15.6% of fasting value). While the minimum fall was 9.4 mg% amounting to 10% of basal value in subject No. 9.

ABNORMAL LIPID LIPOPROTEIN PROFILE IN SUBJECTS OF GROUP IIA

Considering the criteria of labelling an abnormal post prandial response (rise of STC and/or LDL with or without fall of HDL). 3 subjects(No. 2, 6, 8) were found to have unfavourable lipid lipoprotein profile. All these 3 cases showed a rise of STC as well as LDL with little change in HDL at first hour. Thus 3 out of total 9 patients of myocardial infarction (33.33%) had abnormal lipid profile as judged from their post prandial responses.

However, subjects No. 2,3,4,5 exhibited abnormal fasting lipid lipoprotein profile as has been shown in table 15.

TABLE 15 : This reflects that 44% of patients of myocardial infarction had abnormal lipid profile on basis of fasting lipid parameters (mg%).

Sl. No.	Subject No.	Fasting values			
		STC	HDL	LDL	LDL/HDL Ratio
1.	2	226	58	134	2.31
2.	3	225	56	133	2.37
3.	4	236	36	164.8	4.57
4.	5	280	56	184.8	3.30

All these subjects had fasting levels between 226 and 280 mg% with mean level of 241.7±22.4 mg%. Their LDL/HDL ratio varied between 2.31 to 4.57 with mean of 3.13±0.91. This subject No. 2 had both fasting as well as post prandial lipid lipoprotein profile abnormality.

CHANGES IN GROUP IIB

This group was taken from the patients who complained of chest pain. These patients when were subjected to investigations (resting ECG and other relevant investigations) did not reveal any abnormality. Subsequently these patients were subjected to stress ECG, on peak exercise all of them showed ischemic changes. total number of patients studied in this were 9.

CHANGESI IN LIPID LIPOPROTEIN PROFILE

I(a) : CHANGES IN STC

The fasting STC level was below 200 mg% in 3 subjects (33%) and was above 200 mg% in rest of 6 subjects (67%). The STC level increased at first post prandial hour in 4 subjects(44%), while it decreased below fasting level in remaining 5(56%) subjects. The mean STC in this group of patients was 209.5±18.3 mg%, which increased to 212.4±20 mg% at first hour. The mean value remained almost same at third hour (212.4±17.0 mg%). These changes were statistically insignificant (Table 16).

TABLE 16 : Showing changes in STC after HCFD in group IIB (Mean±S.D. mg%).

Fasting (I)	1 hour (II)	3 hour (III)
209.5±18.3	212.4±20.0	212.4±17.0
I : II	't' = 0.312	
I : III	't' = 0.339	

The maximum rise in STC at first hour was of 30 mg% (14.7% of basal value) in subject No. 2, while the minimum rise was observed in subject No. 5(2 mg%), 0.4% of fasting value). The maximum fall in STC was observed in subject No. 6. The value fell by 16 mg% from fasting value of 216 mg%(7%). The minimum fall in STC was only 4 mg% (2.06% of fasting value) observed in subject No. 9.

I(b) : CHANGES IN HDL

HDL level increased after feeding in 6 subjects (67%), while it fell in 3(33%) subjects. The mean fasting HDL in this group of patients was 42.6±8.3 mg%. It increased to 48.6±6.1 mg% at first hour. There was slight fall in mean value at third hour (47.7±6.2 mg%). All these changes were statistically insignificant(Table 17).

TABLE 17 : Showing changes in HDL after HCFD in group IIB (mean ±S.D. mg%).

Fasting (I)	1 hour (II)	3 hour(III)
42.6±8.3	48.6±6.1	47.7±6.2
I : II	't' = 1.70	
I : III	't' = 1.43	

The maximum rise in HDL was in subject No. 8 (20 mg%, 52% of fasting value) while the minimum rise was in subject No. 2(8 mg%, 2% of fasting value). The maximum fall was observed in subject No. 3 and 7. Both of them showed a fall of 8 mg%, 16.6% and 13.7% of fasting value respectively. The minimum fall was observed in subject No. 9. It consisted only 2 mg% amounting to 3.8% of fasting value.

I(c) : CHANGES IN STG

Seven (78%) subjects in this group showed a rise in STG level at one hour after feeding, while remaining 2(22%) subjects showed a fall. The mean STG

of this group was 145.5±18.8 mg%. It increased to 152.3 ±22.1 mg% at first hour while the mean level fell marginally to 147.2±19.8 mg% at third hour. The changes in these values were statistically insignificant as has been depicted in table 18.

TABLE 18 : Changes depicted in STG after HCFD in group IIB (Mean±S.D. mg%).

Fasting (I)	1 hour (II)	3hour (III)
145.5±18.8	152.3±22.1	147.2±19.8
I : II	't' = 0.685	
I : III	't' = 0.182	

The maximum rise of STG at one hour among 7 subjects of this group was seen in subject No. 8 (22 mg%, 13.5% of fasting value). The minimum rise was in subject No. 1(4 mg%, 2.9% of fasting value). The maximum fall of STG was of 18 mg% at first hour in subject No. 7, which amounted to 11.3% of fasting value. The minimum fall was observed in subject No. 6, the value fell by only 2 mg% which consisted of 1.4% of fasting value.

I(d) : CHANGES IN LDL

Three subjects (33%) of this group showed rise in LDL level one hour after feeding, five of them(56%) showed a fall at one hour, while one (11%) subject No. 3 showed virtually no change. The maximum rise was of

21.6 mg%, seen in subject No. 7. It consisted of 15.3% of fasting value. The minimum rise was seen in subject No. 8(5.6mg%, 4.8% of fasting value). The maximum fall was observed in subject No. 6. The value at first hour fell by 33.6 mg%, which amounted to 21.5% of fasting value. The minimum fall was observed in subject No. 9 (4 mg%, 4.4% of fasting value).

The mean LDL value of this group was 137.7± 19.1 mg%. The mean value fell to 133.3±20.3 mg% at first hour after feeding. It showed a rise at third hour (135.7±17.2 mg%). These changes were statistically insignificant as shown in table 19.

TABLE 19 : Showing changes in LDL after HCFD in group IIB (Mean ± S.D. mg%).

Fasting (I)	1 hour (II)	3 hour (III)
137.7±19.1	133.3±20.3	135.7±17.2
I : II	't' = 0.633	
I : III	't' = 0.340	

ABNORMAL LIPID LIPOPROTEIN PROFILE IN SUBJECTS OF GROUP II B

When subjects showing rise of STC and/or LDL at first hour of fedding were groupped as "high risk" 2(subject No. 2 and 8) subjects out of 9, were found to be in this group. That is to say 22% of subjects of this group showed abnormal lipid profile as per their postprandial behaviour.

The incidence of subjects having abnormal lipid lipoprotein profile increased quite significantly, when we studied the fasting profile. 4 subjects of this group had fasting STC above 210 mg% and LDL/HDL ratio more than 2.5 (subjects No. 4,5,6 and 7) as has been shown in table 20.

TABLE 20 : Showing fasting lipid profile in group IIB(mg%).

Sl. No.	Subject No.	Fasting values			
		STC	HDL	LDL	LDL/HDL Ratio
1.	4	236	46	164.0	3.5
2.	5	220	34	156.8	4.6
3.	6	216	32	156.0	4.8
4.	7	230	58	140.4	2.5

This amounted to an incidence of 44% of subjects possessing abnormal fasting lipid lipoprotein profile. The range being (216 to 236 mg%) with the mean STC of these 4 subjects (225.5±7.9 mg%). The mean LDL/HDL ratio of these subjects was 3.8±0.9.

INCIDENCE OF ABNORMAL CHOLESTEROL TOLERANCE TEST IN PROVED CASES OF CAD

In our present study group II consisted of patients of coronary artery disease (CAD). It had 2 subgroups, subgroup A possessing patients of

documented myocardial infarction (ECG and other relevant investigations) and subgroup B having patients of angina pectoris proved by stress electrocardiogram. These 2 subgroups had 33% and 22% of subjects showing abnormal cholesterol tolerance test, respectively. Thus out of total number of 18 patients, 5(27.7%) showed abnormal cholesterol tolerance test.

DISCUSSION

DISCUSSION

In this study we have divided the subjects into two group (Group I and II). Group I consisted of healthy subjects who were further subdivided in group IA and IB on the basis of abnormal postprandial responses (abnormal cholesterol tolerance) to high fat cholesterol diet, while their fasting lipid profiles being normal. Abnormal cholesterol tolerance was taken as post prandial increase in STC and LDL with either a meagre (insignificant) rise of HDL or no rise in HDL.

CHANGES IN GROUP IA

This group (n=10) was labelled as high risk case because all of them showed a rise in STC and LDL with either very marginal rise of HDL or fall of HDL on HCFD at first hour. All parameters (STC, HDL, LDL and STG) were well within normal limit at fasting stage.

CHANGES IN STG

The mean fasting value of this group was well within normal limit (163.4$\pm$30.3 mg%). The range of values (130-215 mg%) corresponded to the range expressed in lipid research clinics, 1980. The mean fasting value

was low in vegetarians(n=7) as compared to non vegetarians (n=3). Similar findings was shown by Sacks et al (1975) and West RD et al, 1968.

As 9 of the total 10 subjects were smokers, a comparative study could not be made.

There was difference in mean value of high fat consumers (n=4) (mean value 186.4±30.8 mg%) and low fat consumers (n=6) mean value (148.2±36.2 mg%) This has also been shown in Framingham study (1977).

Ninety percent of subjects (n=9) were active workers (class IV employees), while only 10%(n=1) was sedentary (junior doctors) so we could not make a group comparison as far as STC was concerned.

One hour after feeding of HCFD (800 mg cholesterol; 24 gms fat) all subjects showed rise in STC, the value increased significantly to 205.8±43.2 mg%. This rise may be because of :

1. Stimulation of endogenous synthesis of cholesterol by huge cholesterol fat load.
2. The early rise (just at first postprandial hour) can be explained by the presence of liquid vehicle(milk)

which facilitated rapid mobilisation of cholesterol fat diet from stomach to gut, as subjects were in fasting stage. Earlier workers(Collen et al, 1969; Stuwart et al, 1954) have also reported immediate postprandial response in adults.

3. Inability of the body (enzymes or liver) to utilise this large amount of cholesterol load.

The maximum increase in STC at one hour was 117 mg%, which amounted to 90% of basal value. This huge quantum of rise in STC can't be explained on the basis of existing literature. The rise in mean STC was 40%. This huge rise is tantamount to more than 1000 mg cholesterol in absolute terms (40x10x3) an amount that is much more than the cholesterol eaten (800 mg).

We propose that dietary cholesterol fat stimulation mobilises cholesterol from different stores (Subintimal pool and macrophages) and is responsible for transient huge rise in STC. Our observations needs to be confirmed on larger sample size.

The STC value at third postprandial hour fell and approached the basal value(183.6±25.3 mg%). This latter fall can be explained on the basis of body's

protective mechanisms which come in role and decrease the elevated blood level by probably stimulating the enzymes to utilize or mobilise the cholesterol from blood to body stores.

CHANGES IN HDL

The value of HDL(mean fasting) was 65.8±18.3 mg%. This value showed very slight change (increase) at first hour (69.2±23.6 mg%). The value fell marginally to 58.4±11.2 mg% at third postprandial hour.

This shows that there was virtually no change in post prandial response as far as HDL is concerned and main rise in STC was provided by LDL, slight increase and latter even decrease in HDL reflects abnormal cholesterol tolerance (high risk) of these subjects as was described earlier.

Inability to rise HDL on giving HCFD may be because of some genetic susceptibility(? enzyme deficiency of these subjects to develop premature and accelerated atherosclerosis as HDL has been a well defined protector to atherosclerosis.

CHANGES IN LDL

The mean fasting LDL value was 82.7±34.2 mg%.

It increased at one hour after HCFD to a mean level of 120±35.6 mg%. This increase was statistically significant. This increase in LDL may be because of following causes :

1. Increased formation of LDL from VLDL and possibly from chylomicron.

2. Defect in receptor mediated endocytosis and digestion by lysozymes within .

3. Defects in scavanger cell system which degrade LDL phagocytes in reticuloendothelial system. This system is thought to function solely to degrade the LDL when lipoprotein reaches high concentration. The value at third post prandial hour was again near the basal value (95.6±23.6 mg%) and this may because of body's compensatory mechanisms which take some time to achieve equilibrium.

CHANGES IN STG

The mean fasting STG in this group was 124.1± 43.4 mg%. It remained virtually the same at first hour after HCFD (122.3±44.8 mg%) and then showed a rise. The level at third hour was 150.6±40.2 mg%. Changes in triglyceride levels were insignificant despite a huge fat load (24 gms). The rise at third hour suggests that there was comparatively slow absorption of fat in these subjects. Why these subjects showed a delayed and small quantum of rise cannot be explained on the basis of existing literature.

<u>RELATION OF EXERCISE ECG AND ABNORMAL POST PRANDIAL BEHAVIOUR ON HCFD IN GROUP IA:</u>

Twenty percent subjects (n=2) showed their exercise ECG to be abnormal. This abnormality was in the form of ST-T changes suggestive of ischaemia. Healthy subjects with positive stress test are not the patients of coronary artery disease but they definitely represent a subgroup at higher risk of developing CAD as has been suggested by Clumming et al (1975) and Allen et al(1980). As these two subjects were having many other risk factors like smoking, high fat consumption, together with the fact that they also exhibited abnormal cholesterol tolerance, the changes of their developing CAD is quite high. Further prospective studies on these subjects should be taken to prove this fact.

As fasting lipid profiles of all these subjects were normal, this suggests that if we consider merely fasting profile for screening susceptible individual among healthy population, a substantial number of subjects prone to develop CAD would be missed.

A significant number (30% of this healthy group with abnormal cholesterol tolerance could not achieve target heart rate because of practical difficulties. Had

all subjects have attained target heart rate, the incidence of positive cases would have been much higher. This implies that the risk of CAD among healthy individuals showing abnormal cholesterol tolerance (though having normal fasting lipid profile) may be quite higher than we have observed in our study.

So, we propose that healthy individuals with normal fasting lipid profile must be subjects to HCFD in order to label them high and low risk. Otherwise we would miss many susceptible individuals. We differ from many established studies on this issue which take only fasting hypercholesterolemia and HDL/LDL ratio as risk factors for coronary artery disease (Framingham's heart study update, 1986, Carlson et al, 1985).

CHANGES IN GROUP IB

This group (n=4) was labelled as low risk as all subjects in this group showed a normal cholesterol tolerance i.e. there was neither an increase in STC nor in LDL after HCFD. HDL, also showed virtually no change after feeding.

CHANGES IN STC

The mean fasting value was 148.7±27.8 mg%, the

value fell at one hour to 124.5±22.9 mg%. What causes this fall in STC at just first post prandial hour is not very clear, but we think that LDL receptor mechanism is responsible for this. According to Joseph et al (1982) after an over night fast, there occurs suppression of LDL receptors. We propose that when cholesterol fat load is given after an over night fast these receptors are stimulated in anticipation of cholesterol load that will enter the circulation.

So large amount of LDL from the intravascular compartment shifts into the subendothelial pool reacting in an acute fall of LDL and STC at one hour.

The STC level starts rising again at third postprandial hour (133±22.9 mg%). This slow increase of cholesterol after 3 hours may be because of the absorption of cholesterol and the reverse movement of the LDL that had entered the circulation earlier. We propose that this mechanism works in majority of healthy individuals with normal cholesterol tolerance.

<u>CHANGES IN HDL</u>

The mean fasting HDL in this group was 48.2± 7.9 mg%. This value showed all most no change of HCFD. Similar trend i.e. no significant change in HDL was

also observed in group IA high risk subjects.

CHANGES IN LDL

The mean fasting LDL value of this group was 76.7±22.2 mg%. It fellto 57.5±7.6 mg% at first post prandial hour. The proposed mechanisms of such a fall has already been discussed under changes in STC(LDL receptor mechanism, suggested by Joseph et al). The level started rising at third hour (62.1±13.6 mg%). The possible explanation of which was also discussed.

CHANGES IN STG

The mean fasting STG level was 130±3.6 mg%. It increased to 142±24.8 mg%. The cause of this insignificant rise remains unclear. The level started falling at third post prandial hour.

RELATION OF EXERCISE ECG AND NORMAL POST PRANDIAL BEHAVIOUR ON HCFD IN GROUP IB

Thus all subjects in this group showed a normal cholesterol tolerance - no rise in STC, LDL and HDL. That is why they were labelled as low risk subjects. When these subjects were subjected to stress ECG no one of them revealed any abnormality suggestive of ischemia.

We cannot draw a firm conclusion from this group of the sample size of this group was small(n=4).

We propose that for the trials on healthy individuals with normal cholesterol tolerance (as our group IB subjects) as needed to draw a conclusion. But it gives thurst to our concept that normal cholesterol tolerance is necessary for labelling a subject as low risk or protected, rather than only categorising them on the basis of fasting levels.

In this particular group (IB) all the four subjects were smoker, but apart from this, they did not possess any other risk factors like obesity, hyper-tension, alcohol intake and diabetes.

CHANGES IN GROUP II

Group II consisted of patients of coronary artery disease. It was divided into 2 subgroups - group IIA(n=9) and group IIB(n=9). Group IIA had all the subjects who had documentary evidence of myocardial infarction, while group IIB was consisted by patients of angina pectoris.

CHANGES IN GROUP IIA

CHANGES IN STC

The mean fasting STC in this group was

211.6±32.6 mg%. Six subjects showed rise after HCFD, while remaining three subjects showed a fall. The rise and fall was within 15-5% of basal value.

This marked variability in response has also been observed by many other workers but on long term feeding (Flynn et al, 1979; Sack, 1983). Why this variability occurs with just single dose feeding in a homogenous group (all patients of myocardial infarction)remains unanswered. The value at first hour(whether in the form of rise or fall) started approaching basal value at third hour in all subjects.

CHANGES IN HDL

The mean fasting value was 51.7±8.2 mg%. Variability was again observed in this paramter. Six subjects showed rise while remaining three subjects showed a fall at first hour. The changes, however, were very small and insignificant. Long term studies by Mahley and associates have shown that cholesterol and fat feeding leads to an increase in HDL and Apo. E, regardless whether STC rises or not in healthy and diseased individuals. It seems that single point feeding has little impact on changes in HDL as

was also pointed out in previous section of our discussion.

CHANGES IN LDL

The mean basal value of LDL was 125.6±32 mg%. The mean LDL value remained the same at first hour after feeding but showed a fall at third hour(102.2±24.3 mg%). The late fall in LDL shows that LDL receptor mechanism (Joseph et al) discussed above, which was quick in healthy individuals, is considerably delayed in patients of myocardial infarction.

We propose that patients of myocardial infarction probably have blunted LDL receptor activity in response to HCFD and this may be responsible for the accelerated atherosclerosis in these patients.

CHANGES IN STG

The mean fasting STG of this group of subgroup was 171.7±17.7 mg%. It increased to 188.5±17.4 mg% at first hour and remained all most same at the third post prandial hour. This was a significant rise in response to HCFD. This increased triglyceride level may be because of increased production of VLDL by the liver to prevent stenosis and possibly because of deficient enzyme system of body could not cope up such tremendous load. Arora

et al (1990) are also of the view that patients of CAD have deficient lipoprotein lipase activity which is responsible for greater and prolonged postprandial triglyceridemia.

ABNORMAL FASTING AND POST PRANDIAL LIPID LIPOPROTEIN PROFILE IN SUBJECTS OF GROUP IIA.

When we studied the fasting lipid lipoprotein profile in their group of patients (M.I.). We found that 4 subjects(2,3,4,5) out 9(44%) showed high cholesterol levels, though LDL/HDL ratio was near normal in half of them. Rest two of them showed an unfavourable ratio (more than 3).

When postprandial behaviour was studied. We found that only 3 subjects (3/9) (nos.2,6,8), showed an abnormal cholesterol tolerance in the form of rise of STC and LDL with little change in HDL. Thus incidence of abnormal postprandial lipid profile was 33% in patients of MI. Remaining six subjects showed either a fall in STC and LDL(4/6) or only slight rise in these parameters(2/6). HDL again showed little change after feeding in these subjects.

Relationship between abnormal cholesterol level and early and accelerated atherosclerosis has been demon-

strated many years back. Anitsihkow et al and Beveridge et al (1959) showed that large amount of cholesterol not only produces hypercholesterolemia but also vascular lesion similar to atherosclerosis in experimental animals. Many workers have reported relativelyi high incidence of hypercholesterolemia in patients of myocardial infarction, though exact incidence in different series has not been mentioned.

CHANGES IN GROUP IIB

CHANGES IN STC

The mean fasting value was 209.5±18.3 mg%. It increased to 212.4±20 mg% at first hour and remained almost same at third hour. The variability was again observed in this group of patients of angina pectoris as five out of nine (5/9) showed fall at first hour, while four showed increase. This behaviour has already been discussed under heading changes in STC in group IIA.

CHANGES IN HDL

The fasting value was 42.6±8.3 mg%. It increased to 48.6±6.1 mg% at first hours and remained almost same at third hour (47.7±6.2 mg%). Thus the post prandial behaviour of HDL has been virtually same through out the study

out the study i.e. little change after single dose of HCFD.

CHANGES IN LDL

The mean fasting value was 137.7±19.1 mg%. It decreased marginally to 133.3±20.3 mg% at first hour and increased marginally to 135.7±17.2 mg% at the third hour. In contrast to patients of MI where a late significant fall in LDL was observed, patients of angina pectoris showed little variation in LDL. The variability was observed in form of rise in 3 subjects (3/9) insignificant fall in 5 subjects (5/9) insignificant and virtually no changes in one subjects (1/9).

The proposed mechanisms for fall in such patients has already been discussed, but why some of these patients showed rise remained to be explored.

CHANGES IN STG

The mean fasting value of STC in this group of angina pectoris was 145.5±18.8 mg%. It increased insignificantly to 152.3±22.1 mg% at one hour and then fall to 147.2±19.8 mg%. These all changes were insignificant when the whole group was considered. But (7/9) subjects showed a significant rise in STG at one hour, an observation

which was similar in patients of MI. Remaining 2 subjects showed a fall in STG. The reason of this fall is not clear.

The proposed explanation for rise of STG has already been outlined in healthy changes in STG in group IIA.

ABNORMAL FASTING AND POSTPRANDIAL BEHAVIOUR ON HCFD IN SUBJECTS OF GROUP IIB

When fasting lipid lipoprotein profile were studied, 4 subjects (No. 4,5,6, & 7) out of 9(4/9) were found to have elevated STC (more than 215 mg%) and LDL/HDL ratio of more than 2.5. This amounted to an incidence of 44%.

On observing post prandial behaviour only 2 subjects (No. 2 and 9) showed an increase in STC and LDL with virtually no change in HDL i.e. abnormal cholesterol tolerance. Thus the incidence comes out to be 22%.

INCIDENCE OF ABNORMAL CHOLESTEROL TOLERANCE IN PATIENTS OF CAD (GROUP II)

This group, which had both patients of myocardial infarction (n=9) and angina (n=9) showed a total incidence of 28% (5/18) of patients showing abnormal cholesterol tolerance.

SUMMARY AND CONCLUSION

SUMMARY AND CONCLUSIONS

The present study was carried out in 32 male subjects aged 40-60 years. These were divided into two groups. (Grou I and group II). Group I consisted of 14 healthy males, while group II consisted of 18 patients of CAD(both M.I. and angina pectoris). Group I was further divided into two groups (IA and IB) on the basis of post prandial response to HCFD as high and low risk. Subjects of group I were subjected to stress ECG while those of group II were analysed for fasting and postprandial lipid lipoprotein profile after HCFD. Observations regarding ECG abnormalities suggestive of ischaemia in group I and lipid lipoprotein profile abnormalities (both fasting and post prandial) in group II were made and statistically analysed.

Following conclusionswere drawn on the basis of present study.

1. Among healthy subjects who were labelled as high risk on the basis of postprandial responses, 20% showed ECG abnormality in ischaemic changes on stresss exercise.

3. ECG abnormality was found in those healthy subjects who were smokers, obese and sedentary workers.

3. Subjects who showed normal cholesterol tolerance (low risk) did not reveal any ECG abnormality.

4. 44% (4/9) patients of myocardial infarction showed abnormal fasting lipid lipoprotein profile while 33%(3/9) showed abnormal cholesterol tolerance to single dose HCFD.

5. When patients of angina pectoris were analysed for fasting and postprandial lipid lipoprotein. profile, 44%(4/9) of them showed abnormal fasting lipid profile, while only 22%(2/9) showed an abnormal cholesterol tolerance to HCFD.

6. When incidence of abnormal fasting and postprandial lipid lipoprotein among patients of CAD (MI and angina pectoris) combindly were analysed, it come out to be 44%. When fasting profiles were studied and 28% when post prandial behaviour was observed.

B I B L I O G R A P H Y

BIBLIOGRAPHY

1. Albrink KJ and Man EB : Serum triglyceride in coronary artery disease. Arch. Intern. Med., 103 : 4; 1959.

2. Allen WH, Aronow WS, Goodman P and Stinson P : Five year follow up of maximal treadmill stress test in asymptomatic men and women. Circulation, 62:522;1980.

3. Aronow WS, and Kaplan NM : Smoking : In Kaplan NM and Stamler JS (Eds.) : Prevention of CAD practical management of risk factors. Philadelphia, WB, Saunders Company, p. 51; 1983.

4. Arora RC, Agarwal N, Arora S, Kumar N, Lakhtakia S : Post heparin lipoprotein lipase activity in patients of IHD and controls. JAPI, 38:635; 1990.

5. Barret DW : Alimentary lipemia in man with coronary artery disease and in controls. M.B.J.,2:640;1956.

6. Becker GH, Meyor J, Necheles H : Fat absorption and atherosclerosis. Science, 111:529; 1949.

7. Bhattacharya AK, Conner WE, Mannolf FA et al : Turn over of xanthoma cholesterol in hyperlipoproteinemic patients. J. Lab. Clin.Med.,87:503; 1976.

8. Blum CB, Levy RI, Eisenberg S et al : High density lipoprotein metabolism in man. J. Clin. Invest., 60 : 795; 1977.

9. Boulton TJC : Validity of screening for hypercholesterolemia at different ages from 2-7 years. Aust., N.Z.J. Med., 9 : 542; 1979.

10. Carlson LA and Bottiger LE, Ischaemic heart disease in relation to fasting value of plasma triglyceride and cholesterol : Stockholm Prospective study. Lancet, 1 : 865; 1972.

11. Carlson LA and Ericsson M : Quantitative and qualitative serum lipoprotein analysis. Part I in healthy men. Atherosclerosis, 21 : 417-443; 1975.

12. Carlson LA and Bottiger LE : Risk factors for ischemic heart disease in men and women. Acta. Med. Scand., 218 : 207; 1985.

13. Castelli WP, Garrison RJ, Dawber TR; Mac Wamara PM, Feinleib M and Kannel WB : The filter cigarette and CHD. The Framingham study. Lancet, 2:109; 1981.

14. Castelli P : Framingham heart study update : Cholesterol triglycerides, lipoproteins and the risk of coronary heart disease. Perspect. Lipid. Dis., 3 : 20 ; 1985.

15. Conner WE, Hodge RE and Bleiler RE : Serum lipids in men receiving high cholesterol and cholesterol free diet. J. Clin Invest., 40 : 894; 1961.

16. Conner WE and Conner SL : Dietary treatment of hyperlipidemia. In Rifkind BM and Levy RI (Ed.) : Hyperlipidemia : Diagnosis and therapy. New York, Grune and Stratton, 1977.

17. Cumming GR, Samm J, Borysy KL and Kich : Electrocardiographic changes during exercise in asymptomatic men. Three year follow up. Can. Med. Asso. J., 112 : 578; 1975.

18. David F, Brown A, Sendra Heslin and Joseph T Doyle : Post prandial lipemia in health and in ischemic heart disease. New Engl.J.Med.,264:15; 733-737; 1961.

19. Froelcher VF, Jr. Thomas MM, Pillow C and Lancaster MC: Epidemiology study of asymptomatic men screened by maximal treadmill testing for latent coronary artery disease. Am. J. Cardiol., 34 : 770; 1974.

20. Geer JC and Haust MO : Monographs on atherosclerosis. S. Karger. Basal, Switzerland Vol. 2 p. 1; 1972.

21. Glueck CJ : Relationship of lipid disorders of coronary heart disease. Am. J.Med., 74:10; 1983.

22. Gordon T, Castelli WP, Hjorland MC et al : High density lipoprotein as a protective factor against coronary heart disease. The Framingham study. Am. J. Med., 62 : 707; 1977.

23. Hanno Krauss Pieter Groot, Ellen Van Ram Shorst et al: chylomicron metabolism in coronary atherosclerosis. Circulation Suppl. Part II. Vol.76 No. 4, Oct.,1987.

24. Hartung GH, Foreyt JP, Mitchell JG, Reeves RS and Gotto AM Jr. : Effect of alcohol intake on HDL cholesterol levels in runners and inactive men. J.A.M.A., 249 : 747; 1983.

25. Herzstein J, Wang C, Adlersberg D : Fat loading studies in relation to age. Circulation,8:450;1953.

26. Kannel WB : Hypertension, blood lipids and cigarette smoking as a co-risk factors for CAD. N.Y. Acad., Sci., 304; 128; 1978.

27. Kannel WB, Castelli WP, Gordon T : Cholesterol in the prediction of atherosclerotic disease. Ann. Intern. Med., 90 : 85 : 1979.

28. LaRosa JC, Cleary and Muesing RA : Effect of long term moderate physical exercise in plasma lipoprotein The natural exercise and heart disease and project. Arch. Intern. Med., 142 : 2269; 1982.

29. Lipid research clinic programm. The lipid research clinics coronary prevention trial results I. Reduction in incidence of coronary heart disease. II. The relationship of reduction in incidence of coronary heart disease to cholesterol lowering. J.A.M.S., 251 : 351; 1984.

30. Mahley RW : Alterations in plasma lipoproteins induced by cholesterol feeding in animals including man. In dietachy JN, Gotto ANJr. and Ontho JA Eds. Disturbances in lipid and lipoprotein metabolism. Bethesda. Amer. Physiol. Soc., 181-197; 1978.

31. McGee DL, Reed DM, Yano J, Kagan A and Yelloston J: Ten year incidence of CAD in Honolulu heart program. Am. J. Epidemiol, 119 : 667;1984.

32. McGill JC Jr. : Atherosclerosis. Problem in pathogenesis. Atherosclerosis Rev., 2:27; 1977.

33. Miller NE : Coronary atherosclerosis and plasma lipoproteins : J. Cardiovasc. Pharmacol. (Suppl 2) 4 : 190; 1982.

34. Nichaman MZ, Sweelay CC and Olson RE : Plasma fatty acids in normolipidemic and hyperlipemic subjects during fasting and after linoleate feeding. Am. J. Clin. Nutr., 20 : 1057 ; 1967.

35. Rose G : Familial patterns in ischemic heart disease. Br.J. Prevent. Soc. Med., 18:75; 1960.

36. Rose R, and Glomset JA : The pathogenesis of atherosclerosis. N. Eng. J. Med., 295; 369; 1976.

37. Ross R and Harper L : Hyperlipidemia and atherosclerosis. Science, 193 : 1094; 1976.

38. Shapiro S, Weinblatt E, Frank CW, Sager RV : Incidence of CAD in a population insured for medical care (HP) : Myocardial infarction, angina pectoris and possiblemyocardial infarction.
Am. J. Publ. Health, 59 (Suppl.), 1 : 1969.

39. Shekelle RB, Schryrock A, Paul O, Lepper M, Stamler J, Liv S and Raynor WJ Jr. : Diet, serum cholesterol and death from coronary heart disease. The Western electric study. N. Eng. J.Med., 304 : 65; 1982.

40. Shepherd o#, Packard CJ, Patsch JR et al : Effects of dietary polyunsaturated and saturated fat on the properties of high density lipoprotein and the metabolism and apolipoproteins A-1. J. Clin. Invest., 61 : 1582; 1978.

41. Taylor CB, Cox GE, Counts M et al : Fatal myocardial infarction in rhesus monkeys with diet induced hypercholesterolemia. Circulation, 20 : 975; 1959.

42. William E, Connor RE, Hodges Roberts E, Bleher : Effect of dietary cholesterol upon serum lipid man.

MASTER CHART

MASTER - CHART

PHYSICAL CHARACTERISTICS OF SUBJECTS

Sl. No.	Name	Age (years)	Sex	Height (cms)	Weight (kg)
		GROUP IA			
1.	Mr. Dhani Ram	42	Male	162	68
2.	Mr. Raman Gupta	30	Male	164	66
3.	Mr. Ram Sewak	46	Male	159	62
4.	Mr. Munshi Lal	60	Male	157	64
5.	Mr. Vijay Singh	40	Male	160	58
6.	Mr. Ram Singh	48	Male	159	54
7.	Mr. Sarman P.	40	Male	163	64
8.	Mr. Kishori Ram	46	Male	160	58
9.	Mr. Kashi P.	48	Male	161	62
10.	Mr. Saru	47	Male	165	63
		GROUP IB			
1.	Mr. Singh	44	Male	163	62
2.	Mr. Gaj raj	42	Male	159	64
3.	Mr. Babu Lal	46	Male	157	59
4.	Mr. Chander Bhan	47	Male	159	60
		GROUP IIA			
1.	Mr. Dinesh Kumar	58	Male	158	60
2.	Mr. Raj Kumar	52	Male	160	61
3.	Mr. N.R.Rawat	64	Male	159	59
4.	Mr. R.K. Yadav	60	Male	162	62
5.	Mr. L.N. Yadav	43	Male	160	63
6.	Mr. P. Tripathi	50	Male	157	60
7.	Mr. H. Chandra	40	Male	157	61
8.	Mr. Radhey Shyama	44	Male	160	58
9.	Mr. Santosh Kumar	46	Male	161	60
		GROUP IIB			
1.	Mr. S.C. Atri	52	Male	172	60
2.	Mr. S.N. Tripathi	50	Male	160	62
3.	Mr. Sudama	54	Male	156	64
4.	Mr. U.P. Chaubey	46	Male	158	62
5.	Mr. H.S. Sakya	48	Male	160	57
6.	Mr. R.C. Shukla	50	Male	161	60
7.	Mr. Vijay Kumar	43	Male	157	62
8.	Mr. Hari Shanker	44	Male	159	58
9.	Mr. Raj Kumar Saxena	46	Male	158	62

MASTER CHART

Fasting and postprandial lipid lipoprotein profile in group IA subjects.

Sl. No.	STC			HDL			STG			VLDL			LDL		
	F	I	III	F	I	III	F	I	III	F	I	III	F	I	III
1.	177	216	200	56	60	62	160	165	170	32.0	33.0	35.0	89.0	123.0	118.0
2.	130	145	150	50	47	42	130	110	116	26.0	22.0	23.2	54.0	76.0	84.8
3.	199	222	216	53	38	40	200	220	226	40.0	44.0	45.2	106.0	140.0	130.8
4.	143	262	205	62	72	66	85	100	80	17.0	20.0	16.0	164.0	170.0	123.0
5.	169	273	191	62	113	66	85	60	120	17.0	12.0	24.0	90.0	148.0	100.0
6.	130	247	171	60	111	56	71	100	182	14.2	20.0	36.4	55.8	116.0	78.0
7.	146	183	164	65	59	60	142	120	182	28.4	24.0	36.4	52.6	79.0	63.6
8.	215	221	205	115	66	79	85	80	120	20.0	22.4	32.0	94.0	152.6	100.0
9.	195	247	198	81	72	63	100	112	160	17.0	16.0	24.0	83.0	139.0	102.0
10.	130	142	136	54	54	50	183	156	150	36.6	31.2	30.0	39.6	56.8	56.0

STC = Serum total Cholesterol,
STG = Serum Triglycerides,
LDL = Low density lipoprotein.

HDL = High density lipoprotein,
VLDL = Very low density lipoprotein.

RESTING AND EXERCISE ECG AND OTHER RELATED PARAMETERS IN GROUP IA

Sl. No.	Heart rate/ min.	Blood Pressure mm Hg Recumbent	Blood Pressure mm Hg Standing	Abnormality if any.	Max. Heart rate/min	B.P. at peak exercise	Abnormality if any.	Interpretation
1.	62	128/70	130/72	NAD	130	138/74	NAD	NORMAL
2.	74	150/80	154/80	NAD	168	164/94	NAD	Normal
3.	80	104/70	102/70	Resting ECG shows T in III & V_1	158	136/82	Inverted T wave in III and V_1 became upright	Normal Resting & Stress ECG
4.	60	124/80	130/84	NAD Sinus Bracy cardia	126	136/84	NAD	Normal
5.	115	130/86	130/84	Sinus Tachy cardia	186	190/86	NAD	Normal
6.	65	118/74	118/72	NAD	165	160/90	Inverted T wave in aVL only	Normal
7.	78	136/86	136/86	NAD	163	158/94	Normal	Normal
8.	100	140/80	144/80	Sinus Tachycardia	166	170/84	ST segment depression 8.5 mm from J point with T wave inversion all precordial leads	Abnormal indicates ischemic changes in exercise
9.	90	120/76	124/80	Normal	170	148/90	Normal	Normal
10.	86	136/86	134/90	Normal ECG	168	168/96	ST segment depression of 2 mm from J point (down sloping)	Resting ECG -normal, Stress ECG-Ischemic changes.

Fasting and postprandial lipid lipoprotein profile in group IB subjects.

Sl. No.	STC			HDL			STG			VLDL			LDL		
	F	I	III	F	I	III	F	I	III	F	I	III	F	I	III
1.	112	88	100	41	38	36	130	116	120	26.0	23.0	24.0	48.0	51.0	40.0
2.	141	124	138	47	47	40	133	133	142	26.6	26.6	28.4	67.0	50.0	61.6
3.	147	136	138	37	39	32	133	183	156	26.6	36.6	31.2	83.0	60.0	72.8
4.	195	150	164	68	61	58	124	136	130	24.8	27.2	26.0	108.8	69.0	74.0

RESTING AND EXERCISE ECG AND OTHER RELATED PARAMETERS IN GROUP IB.

Sl. No.	Heart rate/ min.	Blood Pressure mm Hg		Abnor-mality if any.	Max. heart rate/ min.	B.P. at peak exercise	Abnormality if any.	Inter-pretation
		Recumbent	Standing					
1.	68	110/70	110/74	Normal ECGZ	162	136/86	Normal	Normal
2.	92	120/70	120/74	Normal ECG	150	146/90	Normal	Normal
3.	78	130/70	130/70	Normal ECG	150	148/86	Normal	Normal
4.	90	120/84	120/89	Normal ECG	170	170/90	Normal	Normal

Fasting and postprandial lipid lipoprotein profile in group IIA subjects.

Sl. No.	STC			HDL			STG			VLDL			LDL		
	F	I	III	F	I	III	F	I	III	F	I	III	F	I	III
1.	208	220	216	40	56	46	180	196	200	36.0	39.2	40.0	132.0	124.8	130.0
2.	226	242	220	58	64	66	170	200	190	34.0	40.0	38.0	134.0	138.0	116.0
3.	225	210	220	56	60	60	180	194	200	36.0	38.8	40.0	133.0	112.2	94.0
4.	236	230	216	36	26	30	176	186	180	35.2	37.2	36.0	164.8	166.8	120.0
5.	280	260	273	56	50	42	196	208	200	39.2	41.6	40.0	184.8	168.4	126.0
6.	200	226	220	56	50	60	156	178	160	31.2	35.6	32.0	112.8	140.4	86.0
7.	180	196	200	58	64	70	136	152	158	27.2	30.4	31.6	94.8	101.6	50.4
8.	182	206	200	60	68	54	190	210	208	38.0	42.0	41.6	84.0	96.0	114.4
9.	168	176	170	46	60	54	156	173	176	31.2	34.6	35.2	90.8	81.4	83.8
10.	173	-	-	60	-	-	190	-	-	38.0	-	-	75.0	-	-

Fasting and postprandial lipid lipoprotein profile in group IIB subjects.

Sl. No.	STC			HDL			STG			VLDL			LDL		
	F	I	III	F	I	III	F	I	III	F	I	III	F	I	III
1.	180	170	176	36	40	42	136	140	146	27.2	28.0	29.2	116.8	102.0	104.8
2.	206	236	230	40	48	40	166	180	176	33.2	36.0	35.2	132.8	152.0	159.8
3.	218	212	220	48	40	44	106	118	120	21.2	23.6	24.0	148.8	148.4	152.0
4.	236	230	230	46	58	54	130	138	120	26.0	27.6	24.0	164.0	144.4	152.0
5.	220	222	218	34	48	52	146	153	150	29.2	30.6	30.0	156.8	143.4	136.0
6.	216	200	206	32	50	46	140	138	130	28.0	27.6	26.0	156.0	122.4	134.0
7.	230	236	228	58	46	60	158	140	148	31.6	28.0	29.6	140.4	162.0	138.4
8.	186.	216	208	38	58	42	162	184	160	32.4	36.8	32.0	115.6	121.2	134.0
9.	194	190	196	52	50	50	166	180	175	33.2	36.0	35.0	108.8	104.0	111.0